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Uniform Immunization on Malware Spread in Complex Communication Networks

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Abstract

In this paper, we investigate the impact of uniform immunization as a control measure to mitigate the spread of a malware model over communication networks with complex topologies. Particularly, we formulate the system dynamics using mean-field approximation and obtain the minimum fraction of nodes that need to be immunized to prevent an epidemic outbreak. Results obtained through simulations over both, homogeneous and heterogeneous networks reveal the ineffectiveness of the uniform immunization scheme in networks with high heterogeneity.

Ⅰ. Introduction

Complex information networks such as the Internet and the World Wide Web (WWW) play a vital role in our everyday lives. Such networks are distinguished based on features such as their clustering coefficients and average path lengths. Networks are either *small-world* (SW) or *scale-free* (SF) in nature. While SW networks exhibit Poisson degree distribution, SF networks have a power-law distribution. As a result, the heterogeneity of such networks substantially influences the manner in which spreading processes such as computer viruses and botnets propagate over them [1]. Studying the dynamical behavior of such processes over complex networks is important so as to predict and prevent severe economical and human losses.

Most existing works model the behavior of malwares using compartmental differential equations using mean-filed theory [2]. These works however, mainly focus on fundamental epidemic models such as the *susceptible-infected-susceptible* (SIS) and the *susceptible-infected-recovered* (SIR) themes. Nonetheless, in real world, multipartite malwares may not only infect nodes in many stages, but also exploit mediums such as email attachments and file sharing to spread. Taking these two factors into account, we consider the refined SIS model reported in [3] for our study. For malware models, techniques have also been designed to alleviate the possibilities of and epidemic outbreak. One such technique is known as uniform immunization in which a fraction of nodes are immunized in the network according to a uniform distribution. The potency of this method heavily depends on the nature of the malware model and the underlying network topology. To our best knowledge, there exists no analytical work on the impact of uniform immunization strategy on the model in [3]. To this end, we present mathematical and simulation results to evaluate the uniform immunization strategy on the malware model over both, SW and SF complex networks.

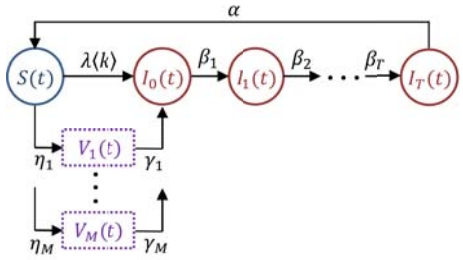

Fig.1: Malware model with infection delay and vectors [3].

Ⅱ. Mean-field Formulation

The malware model of Figure 1 is made up of three types of compartments (or classes), where $S(t)$, $I_i(t)$, and $V_j(t)$ denote the fractions of susceptible nodes, nodes in the i^{th} stage of infection $(i = 0,1,\dots,T)$, and the j^{th} infective vector $(j = 1,2,\dots,M)$ at time t, respectively. With $\langle k\rangle$ as the mean network degree, the remaining notations refer to transmission rates from one particular compartment to another. In addition, we assume a fixed network size with $\sum_{r=0}^{T} I_r(t) + S(t) = 1$. To show the impact of uniform immunization, we henceforth use g to denote the density of immunized nodes in the network.

A. Small-world Homogeneous Networks

In SW networks, the degree distribution is homogeneous, i.e. the degree of every node in such a network is almost the same as the average degree $\langle k\rangle$. By considering Watts-Strogatz (WS) model of SW networks, the system is expressed as follows [3]:

$$\begin{cases} I_0'(t) = -I_0(t) + \lambda\langle k\rangle[1-\rho(t)]\,\rho(t) + \sum_{j=1}^{M} \gamma_j[1-\rho(t)]V_j(t) \\ I_i'(t) = \beta_i I_{i-1}(t) - I_i(t)\,; \quad i = 1,2,\dots,T \\ V_j'(t) = -V_j(t) + \eta_j\big[1-V_j(t)\big]\,\rho(t)\,, \end{cases} \tag{1}$$

where $\rho(t) = \sum_{i=0}^{T} I_i(t)$. Replacing λ and γ_j with $\lambda(1-g)$ and $\gamma_j(1-g)$, respectively, and solving system (1) yields the minimum fraction of nodes that must be immunized in order

to prevent the malware infection from becoming an endemic. This minimum fraction, g_c, should satisfy the following:

$$\left(\lambda \langle k \rangle + \sum_{j=1}^{M} \eta_j \gamma_j\right)(1 - g_c)\, B - 1 = 0\,, \tag{2}$$

where $B = (1/\alpha + 1/\beta_1 + \cdots + 1/\beta_T)$. The expression for g_c is a monotonously increasing function of variables λ, $\langle k \rangle$, η_j and β_i and is given as follows:

$$g_c = 1 - \frac{1}{\left(\lambda \langle k \rangle + \sum_{j=1}^{M} \eta_j \gamma_j\right) B}\,. \tag{3}$$

B. Scale-free Heterogeneous Networks

Unlike SW networks, the node degrees in SF networks are less likely to be similar to the average degree $\langle k \rangle$. A famous SF network model is the Barabási-Albert (BA) model that has a power-law degree distribution and is built upon the notion of preferential attachment. As given in [3], we have:

$$\begin{cases} I'_{k,0}(t) = -I_{k,0}(t) + \lambda k[1 - \rho_k(t)]\,\Theta(t) \\ \qquad\qquad + \sum_{j=1}^{M} \gamma_j [1 - \rho_k(t)] V_j(t) \\ I'_{k,i}(t) = \beta_i I_{k,i-1}(t) - I_{k,i}(t)\,; \quad i = 1,2,\ldots,T \\ V'_j(t) = -V_j(t) + \eta_j \left[1 - V_j(t)\right] \Theta(t)\,, \end{cases} \tag{4}$$

with $I_{k,i}(t)$, $\rho_k(t)$, and $\Theta(t)$ as the infected fraction having degree k and in infection stage i, sum of infected nodes in all stages, and the probability of linking to an infected node at time t, respectively. Following the same procedure as for SW networks, we derive g_c for SF networks such that:

$$\frac{(1-g)B}{\langle k \rangle}\left(\lambda \langle k^2 \rangle + \langle k \rangle \sum_{j=1}^{M} \eta_j \gamma_j\right) = 1\,, \tag{5}$$

with $\langle k^2 \rangle$ as the variance of network degree. The expression for g_c is thus derived as follows:

$$g_c = 1 - \frac{\langle k \rangle}{\left(\lambda \langle k^2 \rangle + \langle k \rangle \sum_{j=1}^{M} \eta_j \gamma_j\right) B}\,. \tag{6}$$

Ⅲ. Simulation Results and Discussions

To validate our analytical results, we simulate a network of 1000 nodes with $\langle k \rangle = 6$, $\lambda = 0.07$, $\beta_i = \eta_j = \gamma_j = \alpha = 0.2$ and consider only one infective medium for simplicity.

Figure 2 depicts the minimum immunized fraction of nodes as function of the infection rate λ. Note that for any fixed λ value, g_c increases with the number of infection delay stages from $T = 0$ to $T = 3$. Yet, in a BA network, g_c instantly shoots up for very small values of λ as illustrated in Figure 3. Comparing the two network models under same parametric settings, we see that the sudden rise of g_c in BA networks is due to the very low epidemic threshold inherent in such networks. In other words, due to the high heterogeneity in connectivity properties of SF networks, they tend to have a very low critical threshold for epidemic outbreak and thus, show weakness in face of malware spread. Thus, we conclude

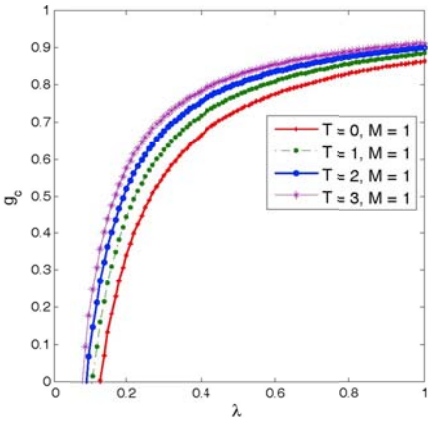

Fig.2: Immunized fraction (g_c) versus infection rate (λ) in a homogeneous network.

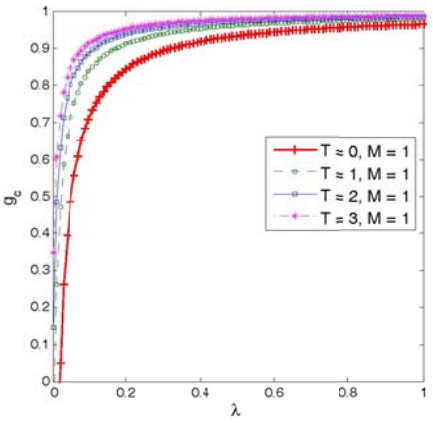

Fig.3: Immunized fraction (g_c) versus infection rate (λ) in a heterogeneous network.

that uniform immunization is not effective in SF networks due to their peculiar hierarchical structure, but can be used in SF networks to reduce the impact of malware spread. These results clearly imply the need for further investigation of better immunization strategies over SF networks on account of their unique topological characteristics.

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